Cognitive-Behavioral Therapies for Trauma

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C H A P T E R S E V E N

Anger and Trauma

CONCEPTUALIZATION, ASSESSMENT, AND TREATMENT

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The activation of anger has long been recognized as a feature of clinical disorders that result from trauma, such as dissociative amnesia, dissociated identity, borderline personality, and brain-damage dementia. However, attention to the involvement of anger has occurred most notably with respect to posttraumatic stress disorder (PTSD), for which anger is considered a salient attribute of the arousal symptom cluster. Within the PTSD field, in both clinical and research contexts, concern with anger has occurred primarily in conjunction with combat-related PTSD. Therefore, much of this chapter will pertain to that domain.

The literature pertaining to anger and trauma begins with some classic works in the trauma field. We connect these to contemporary research that points to anger as an important clinical and theoretical variable. We present our own theoretical orientation, which is a regulatory deficits model, and describe types of anger dysregulation. Procedures for anger assessment are discussed, and their congruence with measures of PTSD are highlighted. Anger-focused cognitive-behavioral therapy is an important adjunctive treatment for PTSD, and an approach to anger treatment is presented. A recent, controlled clinical trial that produced significant treatment gains with patients having severe anger and severe PTSD will be discussed. Recommendations are offered for implementing anger treatment, being mindful of the treatment-resistant characteristics of this patient population.

ANGER AS A REACTION TO TRAUMA: EARLY OBSERVATIONS

Anger has long been identified as a component of traumatic reactions. Freud's writings about affect associated with trauma largely ignored anger. Although he did say some things about retroflected anger in *Mourning and Melancholia,* Freud was preoccupied with fear and anxiety as traumatic affects. However, important historical work in the trauma field, such as Lindeman (1944), Grinker and Spiegel (1945), and Kardiner and Spiegel (1947), offered many observations about anger, hostility, and aggression as trauma-linked responses. The latter two works, which dealt with combat aftereffects, were particularly incisive in describing anger symptom patterns that were given more concerted attention by Vietnam era scholars, such as Bourne (1970), Horowitz and Solomon (1975), and Figley (1978).

Lindeman's analysis of the acute grief observed among patients variously bereaved found irritability and anger to occur along with a disconcerting loss of warmth in personal relationships. He described the hostile reactions as surprising, inexplicable, and disturbing to the patients and perceived by them as "signs of approaching insanity" (p. 142). In addition to these aspects of anger being a regular part of acute grief, he reported that more intense anger occurs in what he termed "distorted reactions" in morbid grief syndromes. There, on top of generalized hostility in social relationships, he found that "furious hostility against specific persons" may emerge, and among the targets for such was the therapist. This latter point is incisive, as anger directed at clinicians indeed represents a significant challenge in anger treatment work.

Early research on combat stress identified anger reactions as central aftereffects. The American soldier drew attention during World War II, because theaters of battle were naturalistic, albeit cruel, domains for the study of psychological trauma and adaptation to extreme environments (Stouffer, 1949). Unmistakably, research on human stress received a key impetus from investigations of psychological functioning in warfare. Grinker and Spiegel (1945) described eruptive anger occurring among flight crewmen reacting to the strains of air combat operations and specified anger and aggression as elements of stress disorder. In the earliest work on psychopathology resulting from combat, Kardiner and Spiegel (1947) described the tendency to aggression and violence as being one of the most common symptoms of traumatic neuroses:

> The aggression may show itself in the tendency to "tempers." Easily aroused to anger, these patients are very prone to motor expression. They either break or tear objects in these fits of temper, or strike the people who happen to be around them. This symptom is subject to wide variations. If the outburst is accompanied by loss of consciousness, the patient is usually dangerous. Often these patients injure themselves unintentionally. . . . The aggressiveness of the traumatic neu-

> rotic is not deliberate or premeditated. His aggression is always impulsive. . . . The sadomasochistic complex is related to the irritability, the incapacity to analyze stimuli in the environment. (pp. 212–213)

Explosive irritability and unwarranted rage were identified by Kardiner and Spiegel as a stage in what they saw as a progressive development of incapacitating breakdowns, beginning with poor appetite and carelessness, then involving irritability and exaggerated reactions of rage, and finally culminating in freezing, sleep disturbances, and being terrified of one's own artillery. What they describe in the previous account of anger and aggression fits our own conception of anger and PTSD developed later in terms of context-inappropriate "survival mode" functioning.

With the Vietnam war, conceptions of combat stress improved from the earlier notions of "shell shock" and "combat fatigue." Stressor conditions were understood to be more than the harsh physical circumstances of war and to include the psychological ambience of combat. All wars involve immersion in a hostile atmosphere, but the clandestine nature of the fighting in Indochina exacerbated the psychological strain. American troops developed "a sense of helplessness at not being able to confront the enemy in set piece battles. The specter of being shot at and having friends killed and maimed by virtually unseen forces generated considerable rage which came to be displaced on anyone or anything available" (DeFazio, 1978, p. 30). Advances in military psychiatry, the general availability of drugs for self-treatment of fear, and the personnel practice of rotations to relative safety provided for coping with acute stress in the combat zone; but it was then observed that veterans manifested "delayed stress responses" (Horowitz & Solomon, 1975), and "indiscriminate rage" emerged as a common theme. Indeed, it was often the experience of rage and the fear of one's destructive impulses that prompted veterans to seek treatment.

Early studies with Vietnam veterans highlighted anger dyscontrol. Figley and Eisenhart (1975) had found that, compared to noncombatant servicemen, combatants got into more verbal fights, more frequently had violent fantasies and dreams, and had fewer close friends. Similarly, DeFazio, Rustin, and Diamond (1975), studying a college sample of Vietnam veterans who had been out of service for an average of 5 years, found that 67% had nightmares and 41% felt themselves to be short-tempered or hotheads. In addition to these hostile impulses and the conjoined fear of loss of self-control, the veterans exhibited pervasive distrust of authority figures, estrangement from society, and considerable contempt for anything connected with the government (Horowitz & Solomon, 1975). Quite poignantly, we found these themes to have remained salient in our recent treatment project involving Vietnam veterans with severe PTSD and severe anger (Chemtob, Novaco, Hamada, & Gross, 1997).

CONTINUING RECOGNITION OF ANGER IN TRAUMA RESEARCH

The identification of anger as a reaction to trauma has primarily been elaborated in research on veterans, although it has by no means been confined to that population. Nevertheless, the research on combat-related PTSD has been preeminent. Anger has been described as a significant symptom of postwar adjustment difficulties in epidemiological (Boulanger, 1986; Laufer, Yager, Frey-Wouters, & Donnellan, 1981; Kubany, Gino, Denny, & Torigoe, 1994; Kulka et al., 1988), clinical (van der Kolk, Boyd, Krystal, & Greenberg, 1984), and laboratory reports (Chemtob, Hamada, Roitblat, & Muraoka, 1994; Lasko, Gurvits, Kuhne, Orr, & Pitman, 1994).

The laboratory research has served to disentangle alternative explanations for observed anger among veterans. In the Lasko et al. (1994) study, veterans with PTSD were found to be significantly higher in anger, hostility, and aggressiveness than non-PTSD veterans. They also found that the group differences in hostility and aggressiveness were not a function of combat exposure, but they did not analyze combat exposure as a covariate of the *anger* measures. Chemtob et al. (1994), in comparing combat veterans with and without PTSD, matched them on combat exposure and included a group of noncombat veterans. Indexed by a multiple measure anger factor, the combat PTSD veterans were significantly higher in anger. Interestingly, these three groups of veterans did not differ in either cognitive or motor impulsivity, and these factors were independent of anger. Moreover, the relationship of anger to PTSD was found to be independent of trait anxiety. Hence, Chemtob et al. demonstrated that combat exposure, impulsivity, and anxiety did not account for the PTSD–anger relationship.

Clinical research has clearly substantiated the relevance of anger in PTSD patients. Veterans with PTSD report more hostility and aggression toward partners than do non-PTSD veterans (Byrne & Riggs, 1996; Carroll, Rueger, Foy, & Donahoe, 1985) and more anger-related job problems (Knight, Keane, Fairbank, Caddell, & Zimering, 1984). Female partners of veterans with PTSD also report having more violence directed toward them and more relationship problems than do the female partners of veterans without PTSD (Jordan et al., 1992). In addition to the harm to others caused by their violent behavior, this tendency toward hostile responding may also put those men at higher risk for cardiovascular disease. Kubany et al. (1994) found that the hostility scores of veterans with PTSD were one standard deviation higher than the mean for an entire group of 1293 veterans, using the Cook–Medley measure that often has been associated with heart disease incidence and mortality. Overall, it has been widely found that anger is a prominent concern for Vietnam veterans seeking clinical services (Blum, Kelley, Meyer, Carlson, & Hodson, 1984; Scurfield, Corker, & Gongla, 1984; Silver & Iocono, 1984). Thus, it has been amply demonstrated that anger and aggression

have wide-ranging impact on veterans, their families, their work settings, and society at large.

The relevance of anger has continued to emerge in trauma research with other populations. Krupnick and Horowitz (1981) found anger to be a major theme in their study groups of trauma cases involving both bereavement and serious personal injury. As evaluated by two clinical judges, "rage at the source" was present in 80% of the bereavement cases and 73% of the personal injury cases. Also "rage at those exempted" was present, respectively, in 40% and 27% of the cases. In terms of the prevalence of the 10 most commonly identified themes, "rage at the source" was second only to "discomfort over vulnerability." In a study by Saunders (1994), involving domestic violence survivors in five states, "irritability and outbursts of anger" occurred as a symptom in 42.5% of the women who obtained help at domestic violence programs and 32.9% of those who obtained help at other types of programs. Indeed, if Saunders's tabled data are examined, "irritability and outbursts of anger" is the fifth highest symptom reported of the 17 symptoms of the DSM-III-R PTSD criteria. Unfortunately, the ordinal position of the anger symptom did not receive comment.

That this relative salience of anger does not receive attention from Saunders is not at all odd, as anger usually has not been a primary investigative concern. In clinical research more generally, anger has typically taken a backseat to depression and anxiety. Thus, it is hardly surprising that the burgeoning research on violence victimization experienced by women standardly ignores anger as a trauma outcome—this can be seen in many studies concerned with domestic violence as a source of PTSD (e.g., Cascardi, O'Leary, Lawrence, & Schlee, 1995; Houscamp & Foy, 1991; Rodriguez, Ryan, Kemp, & Foy, 1997) and in most studies of PTSD as a result of (other) violent crime (e.g., Resnick, Kilpatrick, Dansky, Saunders, & Best, 1993; Resnick, Kilpatrick, & Lipovsky, 1991). Even a superb PTSD treatment study concerning rape victims by Foa, Rothbaum, Riggs, and Murdock (1991) ignored anger as a criterion measure.

In contrast, anger was a quite specific focus of a study by Riggs, Dancu, Gershuny, Greenberg, and Foa (1992) on PTSD among female crime victims. Their investigation concerned 166 victims of violent crime, sexual and nonsexual, and included a comparison group of women who had not been victimized and were matched on age, race, education, and income. The crime victims were further grouped according to PTSD status and were assessed by structured interview and self-report questionnaires (STAXI [State–Trait Anger Expression Inventory] anger and PTSD symptoms) 1 week and 1 month after the assault. While anger was not related to the type of assault (sexual–nonsexual), it was significantly associated with severity of the assault and the victim's responses. Higher anger was found in conjunction with the assailant's use of a weapon and with perceived forcefulness. It was also significantly related to pleading, screaming, or fighting with the assailant. Examining anger as a function of PTSD and victimization, the victim groups had higher state anger than did the nonvictim group, and the victim

groups were significantly distinguished by there being higher anger-in scores for the PTSD group. Riggs et al. speculated that intense anger interfered with the recovery from trauma, as holding in anger may block the modification of the traumatic memory due to the overlap between anger and fear structures.

Anger and trauma have an intriguing relationship. We present our conceptualization of their conjunction and of the dynamics of anger in the context of PTSD. Our model asserts that anger occurs in PTSD as a function of regulatory deficits in cognitive, arousal, and behavioral subsystems. The foundations of this conceptualization are compatible models of anger and of PTSD, which we sequentially describe.

ANGER, AGGRESSION, AND ANGER REGULATION

Anger is an emotional state that has both adaptive and maladaptive effects on behavior. As a normal emotion, it has considerable adaptive value for coping with life's adversities, such as enabling perseverance in overcoming obstacles presented by thwartings and injustice. Because anger is a mobilizing mechanism, it can energize corrective action. It provides for personal resilience and is an important guardian of self-worth. However, because anger can activate aggression, anger control is indispensable for aggression control. In addition to the detriments associated with inducing aggression, anger arousal is also problematic because it can interfere with information processing and thereby impair judgment and problem solving.

Anger activates aggression, but aggressive behavior is not an automatic consequence of anger, because aggression is regulated by inhibitory mechanisms engaged by internal and external factors. Regulatory controls of aggressive behavior, such as external restraints, expectations of punishment, empathy, or consideration of consequences in turn can be overridden by disinhibitory influences; that is, the inhibition of aggression can be overcome by facilitating factors, such as heightened arousal, aggressive modeling, lowered probability of punishment, biochemical agents, and environmental cues (Bandura, 1973, 1983). While research and theory on the regulatory control of aggression are well-established, much remains to be articulated with regard to anger, especially concerning its involvement in clinical disorders such as PTSD.

The relationship of anger to aggressive behavior is that it is a significant activator of aggression and is reciprocally influenced by aggression, but it is neither necessary nor sufficient for aggression to occur (Novaco, 1986). With the exception of some theorists, notably Berkowitz (1990, 1993), it is generally agreed that anger is an activator of aggression (Bandura, 1973; Konecni, 1975; Zillmann, 1979). Berkowitz's contrary view is that anger occurs parallel to aggression and that both are produced by "negative affect" induced by unpleasant external events. Were this to be the case, the association between aggression and depres-

sion would be roughly equivalent to that between aggression and anger, and there is little to substantiate that deduction.

The concept of anger regulation is not addressed systematically by existing theories of anger and aggression. Neither of the two most prominent theories of aggression, those of Bandura and Berkowitz, provide explicit accounts of anger regulation. While such theories do address processes that regulate aggression, they are quite silent about anger. Bandura (1973) clearly stated that aggressive behavior is regulated by environmental cues, which have response-directing functions through the information they carry concerning response consequences. Also, within his general model, arousal activates prepotent responses; therefore, lowering anger arousal will reduce aggression. Regarding the regulation of anger, as distinguished from aggressive behavior, since Bandura viewed external stimuli as having anger-evoking potency though symbolic conditioning, and since he asserted that anger can be self-generated by provocative thoughts, it can be inferred that changes in cognitive meaning systems and in rumination would be expected to have a regulatory effect on anger. Subsequently, Bandura (1983) conceptualized self-regulation as a product of self-reward and self-punishment through the subsidiary processes of self-observation, judgment, and self-response (contingencies applied to oneself). He understands the disengagement of internal control and disinhibition in terms of the resetting of self-evaluative contingencies, such as self-exoneration.

Similarly, Berkowitz's (1993) attention to regulation and control is virtually all given to aggression, only addressing anger regulation in the context of catharsis. Berkowitz (1990) claimed to address anger regulation (given the title of his article), but said surprisingly little about it, only stating that unwanted feelings activate cognitive activity that searches for coping options; that is, awareness of negative feelings prompts thoughts about causes of those feelings and considerations of how to act. His view of anger is that it is associatively linked with the "negative affect" produced by unpleasant events. He regards anger and fear as processes that only parallel the escape and aggressive motor tendencies evoked by negative affect. For Berkowitz, thought only play a small role in the initial stages of anger evocation, as automatic association processes are dominant and govern the initial reactions. He clearly posits cognitive control of aggressive behavior, as when conscious anticipation of punishment can suppress aggression, but for him, cognition has minimal influence on the activation of anger.

Bandura and Berkowitz have primarily sought to understand aggressive behavior and have treated anger as a secondary phenomenon. In contrast, Averill (1982) took anger as his focus, and his social-constructivist approach to emotion conceives of anger as a transitory social role governed by social rules. As his research has concerned normative patterns of anger, his analysis does not provide for an understanding of anger as a clinical problem or a condition of psychological disturbance; that is, he omits dealing with the dysregulation of anger or internal processes that provide for ongoing monitoring of anger states.

Novaco's (1994a) model of anger pertains to its normal and abnormal forms, conceptualizing anger as entailing three reciprocally connected domains: cognitive, arousal, and behavioral. These domains are also linked to an environmental context. Anger is viewed as a product of (1) the cognitive processing of environmental circumstances, (2) conjoined physiological arousal, and (3) behavioral reactions. Novaco's (1994a) model maps these domains of anger and their subdimensions and stipulates their reciprocal connectedness with each other and with environmental circumstances. However, he left regulatory systems unspecified, stating that regarding "the capacity to regulate anger and aggression . . . much remains to be addressed with regard to psychological deficits in anger control" (p. 53).

Guided by the latter model of anger and by the Chemtob, Roitblat, Hamada, Carlson, and Twentyman (1988) information-processing model of PTSD, which posits "survival mode" functioning as a state of dysregulation, Chemtob, Novaco, Hamada, Gross, and Smith (1997) proposed a new model to account for anger in the context of trauma. This new model construes trauma-induced anger in terms of regulatory deficits in cognitive, arousal, and behavioral systems associated with "survival mode" functioning in PTSD. We recapitulate this model in part here.

TRAUMA, ANGER, AND REGULATORY DEFICITS

In conjunction with trauma, anger is intrusive and is part of a dyscontrol syndrome involving heightened arousal, hostile appraisal, and antagonistic behavior activated as a survival response to severe threat. Maladjustment difficulties obviously occur when the intense anger is activated in the absence of genuine survival threat; that is, the traumatized person fails to regulate covert and overt responses in accordance with situational realities.

Chemtob et al. (1988) conceptualized PTSD symptoms as context-inappropriate activation of "survival mode" functioning, which, in the absence of threat, is usually suppressed by normal cognitive processing. "Survival mode" functioning has a number of important features: (1) It is triggered by perceptions of external, life-threatening events or by expectancies about encountering such threats; importantly, threat perception is not always consciously mediated or "thoughtfully" recognized; (2) once triggered, "survival" mode is peremptory and preemptive of other cognitive processing, because dealing with life threats is a superordinate requirement for the organism; (3) it is characterized by specific cognitive biases, including a tendency to give primacy to pattern matching, to require less evidence of threat to engage action, and to be inclined toward threat confirmation and increased vigilance that leads to more efficient recognition of the presence of threat; (4) it entails a substantial load on the organism's capacity to regulate optimally its arousal level, which may lead to impairment in this capacity; and (5) its peremptory quality entails a loss of self-monitoring (i.e., one does not always recognized the shift into a different mode).

In PTSD patients, "survival mode" functioning becomes maladaptive because its activation is routinely incongruent with the state of affairs confronting the individual. This activation can result from several causes, and frequently from dynamic, interrelated causes. For example, associative reminders can serve to provoke cognitive processing into "survival mode." Once this processing is activated, a positive feedback loop and "confirmation bias" (cf. Chemtob et al., 1988) tend to validate the engagement of "survival mode" through the identification of threat-confirmatory aspects of the environment, which in turn increase physiological arousal. This process often escapes top-down regulation due to the experiential urgency that it engenders.

In responding to a threat, real or perceived, the activation of "survival mode" includes the activation of anger structures. Anger schemas are integrated mental representations about environment behavior relationships entailing rules governing threatening situations. For example, rules pertaining to personal protection, response to injustice, challenges to self-worth, and justified retaliation are part of anger schemas, the activation of which includes conjoined arousal and behavioral control information. Because people with PTSD are "primed" to identify threat, they often engage "survival mode" more rapidly. The spreading activation of threat schemas consequently strongly potentiates anger. Conversely, the activation of anger structures can serve to activate the full "survival mode." Importantly, inhibitory controls on aggression can be overridden by the conjoined activation of the hostile appraisal and the heightened arousal.

The linking of anger to survival carries several implications about its activation in PTSD: (1) Its onset carries a coping response urgency that preempts alternative appraisals of the triggering event and considerations of alternative action plans; (2) it engages cognitive processes that dispose or bias the system toward confirmation of the expectation of threat; (3) the association of anger with survival leads to its activation in response to minimal cues; (4) the strong arousal and the peremptory nature of the threat schemas suppress inhibitory controls of aggressive behavior; and (5) threat–anger responses are organized as a positive feedback loop; the more threat is perceived, the more anger and aggression. Conversely, the more anger and aggression, the greater the readiness to perceive the presence of a threat. This self-confirming vicious cycle can be interrupted early in its activation by the detection of disconfirming evidence, including consideration of mitigating circumstances (e.g., such as lack of hostile intent or by trained self-monitoring that permits reframing the episode). However, there is an activation level at which the system triggers into "survival mode" and becomes far more difficult to regulate after the mode shift. Because of the tendency to engage "survival mode" more readily, PTSD patients are more likely to become angry.

Put succinctly, the anger system is viewed as having three major domains (cognition, arousal, and behavior) which are interrelated in activation and inhibition, and it is intrinsically connected to the threat system, with its associated

fear and avoidance responses. Anger regulation is affected by traumatic experience, which resets activation and inhibition patterns in accordance with perceived threat, and by the shift into "survival mode" functioning. Patients with PTSD readily shift into "survival mode," and, as part of the peremptoriness of that shift, there is substantial loss of self-monitoring. The context-inappropriate cognitive distortions, which tend to confirm the presence of threat and lead to the defensive activation of anger and aggression, have for that person a powerful subjective quality of immediacy and validity. A fuller presentation of this conceptual framework can be found in Chemtob, Novaco, Hamada, Gross, and Smith (1997).

Anger dyscontrol is among the most challenging of clinical problems, particularly when it is conjoined with impulsive, aggressive behavior. High-intensity anger combined with diminished inhibitory control is alarming and worrisome. Hence, the occurrence of anger in the context of combat-related PTSD has provoked particular concern about treatment needs and treatment delivery systems. Clinical concerns about anger, however, are not restricted to its potential to activate violent behavior. Riggs et al. (1992), in their study of female crime victims, called for effective treatment of anger, which they viewed as impeding psychological adjustment following trauma, and they speculated that anger during exposure sessions may reduce the efficacy of anxiety treatment.

Because certain clinical populations (e.g., institutionalized psychiatric patients, as well as combat veterans) have had long-standing problems with anger, typically compounded by comorbid substance abuse, instability in employment and personal relationships, and physical health problems, psychotherapeutic treatment of their anger difficulties encounters considerable obstacles. Remediation of anger in such treatment-resistant groups would also sound an optimistic note for anger interventions with other difficult to treat patients, and we have some good news later in that regard.

SPECIAL CHALLENGES IN TREATING ANGRY CLIENTS

Uncontrolled anger, being too easily transformed into destructive aggression, beckons for therapeutic intervention to restore or improve self-regulation. However, the treatment of anger presents a number of special challenges to clinicians and to health care institutions, as the delivery of "anger management" services is less than straightforward. Horowitz and Solomon (1975) described the person with delayed stress syndrome as typically suspicious, easily frustrated, and feeling as though he or she will lose control over hostile impulses. Chronically angry patients are not only treatment resistant, but treating them is also problematic because of their readiness to become angry during therapy and toward the therapist.

Angry people are often fiercely resistant to anger treatment. Because anger can mobilize one's psychological resources, energizing behaviors that take corrective action, the capacity for anger is needed as a survival mechanism. In a world

where the significance of the individual is diminished by bureaucracy, anger is a fortification for a sense of worth. Proposing "anger management" could be viewed disparagingly as an insidious strategy to stifle the individual human personality or to constrain the will to determine one's own destiny. Anger provides for personal resilience. It is a guardian of self-esteem, it potentiates the ability to redress grievances, and it can boost determination to overcome obstacles to happiness and aspirations.

In effect, people can remain attached to anger, because it is so very functional. Dislodging the attachment to anger is a matter of helping the person to see that chronic anger has costs that outweigh the functions that it has been serving. Achieving this hinges on the therapeutic relationship. However, there are a number of refractory impediments to establishing that relationship, as special difficulties arise in the treatment of anger that can thwart or derail the therapeutic process. These challenges or difficulties peculiar to anger treatment are clinician safety, the low frustration tolerance of clients, the instrumentality of anger and aggression, and the resource impoverishment of clients.

Clinician Safety

A prevalent issue for the clinician is personal danger. Indeed, PTSD has been found to be a consequence of patient violence on clinical staff (Caldwell, 1992). Needless to say, it is unsettling for therapists and counselors to work with persons who have explosive tendencies. In cases where there has been a history of violent behavior, the clinician's concern is easily aroused by the client's expressions of anger and by descriptions of anger experiences accompanied by aggressive impulses. It is imperative that the therapist be at ease with client anger expressed in narrative accounts or in direct personal communication in the therapy room or on the ward. Precautions for personal safety should always be in place, particularly in conjunction with treating mentally disordered patients who have been previously violent.

While, on the one hand, anger imparts a sense of mastery, on the other, it can signify that one is out of control. When it attains levels of intense arousal, it can be profoundly troubling to the person having the anger experience. Because of its intrinsic connection to the threat system, strong anger, and its implied loss of control, is anxiety engendering for the client. The admixture of fear can intensify rage reactions. To forestall a spreading activation, the therapist must provide a sense of control and in many ways serve as a role model for how to handle anger experiences. Thus, it is imperative that the therapist not be unduly alarmed by exposure to anger. Remaining calm not only provides a counterbalancing reassurance, it prevents emitting cues that might be read as threat signals in the confirmation-biased perception of the client. Even with regard to anger communications that are relatively low in explosiveness, if the therapist becomes uneasy, the troubled individual might well wonder whether it is safe or the least bit useful to reveal matters of deep personal significance to someone who becomes

unsettled upon hearing the disclosures. When clients sense that their psychological realities alarm the therapist, the helping process is undermined, because their sense of safety is compromised by the therapist's alarm.

Some clients may indeed test the therapist's acceptance of them by describing angry feelings, hostile fantasies, and violent behavior. Distrust often takes the form of anger reactions; hence, composure on the part of the therapist is imperative for enhancing the therapeutic relationship, as well as for clinician safety. Treatment of angry clients requires the mastery of anxiety about assaultive risk. This requires sharpened awareness, safe arrangement of physical surroundings, training in personal protection, and having a security response to crisis.

Low Frustration Tolerance

Persons who are prone to provocation are inherently impatient. Like other types of clients, they are often ambivalent and have poorly defined or unrealistic goals for the course of therapy. They may thus become frustrated when desired treatment effects are not quickly forthcoming. As their frustration mounts, they become inclined to disengage from therapy, the impulse for which may be activated by relatively minor events in their regular life or in conjunction with receiving treatment. Because angry people, by their own long-standing behavior, have raised the probability of exposure to aversive events, the therapist should be prepared for such occasions of client frustration and demoralization.

It is imperative that the clinician exercise good coping skills when faced with client expressions of frustration, viewing this as a manifestation of the clinical problem and to not "take it personally." Rather than making undue personal attributions about the client's reactions, the therapist can utilize the manifest crisis as an opportunity to teach anger coping skills. Instead of merely providing reassurance and attempting redirection, the client's frustration and impatience can be engaged and explored, thereby teaching how to communicate about anger and how to deal with conflict. Beyond the ordinary inertia impeding change, angry patients can feel hopeless about ever being different, particularly if they have been recurrently institutionalized.

Given the impatient disposition of clients with anger problems, it is advantageous for a treatment program to be clearly defined and structured, so as to minimize the frustration that can result from vague expectations regarding treatment. Moreover, the proneness to frustration and impatience that are intrinsic to the problem constellation also dictates that treatment studies be thoughtfully designed with control group conditions that do not activate anger responses.

Instrumentality

Like habitual aggressive behavior, chronic anger is an obstinate problem by virtue of its instrumentality. Anger has considerable value in dealing with aversive

situations, particularly as it imparts a sense of mastery or control. One can overcome constraints and dispatch unwanted others by becoming angry and acting aggressively. Persons who are so disposed are reluctant to relinquish this sense of effectiveness. The propensity for anger reflects a combative orientation in responding to situations of threat and hardship, which is not easily surrendered as a learned style of coping. This has important implications for the clinician's presentation of anger treatment and for maintaining a sense of safety.

If the presentation of "anger control" therapy suggests to clients that their sense of effectance will be jeopardized ("robbed of their power"), then the leverage for treatment is easily undermined. Learning anger control skills must be seen to mean enhancement of effectiveness in handling provocation. Very importantly, "anger control" must be approached in a preventive sense and in an arousal-regulatory sense, as well as with regard to enhancing overt behavioral skills. Clients must learn to ask themselves, not only "What should I do when I get angry," but "How can I not get angry in the first place, and if I do get angry, how can I keep the anger at a moderate level of intensity?" They can be helped to see that, whatever they want to accomplish that is lasting and meaningful, uncontrolled anger does not increase its likelihood of attainment. The costs of unregulated anger are the keystone for therapeutic change.

That anger episodes can be used to coerce desired behavior is sometimes understood in terms of "secondary gain," which we conceptualize as attempts to gain adaptive advantage from disability. With respect to anger, "secondary gain" involves the recognition, conscious or unconscious, that one can manipulate one's own anger state to get others to do what one wants. The key aspects of using anger dysregulation for advantage is to manipulate the fear of aggression, so as to induce others to yield ground. In the context of treatment, therapeutic effectiveness in addressing this special difficulty hinges on the clinician safety theme, as well as management of the countertranferences that angry patients provoke. The latter issue highlights the importance of having procedures for case supervision.

Resource Impoverishment

A fourth impediment to treatment is that clients with problems of anger and aggression have deficiencies in cognitive, social, and economic resources. Persons institutionalized for mental disorder, developmental disabilities, or criminal behavior are often of low socioeconomic status (SES) and have few resources to overcome their anger difficulties, and as we noted at the outset, combat veterans with PTSD have had significant postwar adjustment difficulties in the realms of employment, family, and health. Several dysfunctional reciprocities between anger/aggression and resource variables exacerbate treatment difficulties.

Eruptions of anger are not conducive to job stability. Reciprocally, unstable employment raises the risk of anger and aggression by increasing aversive expe-

riences and other motives for aggression, as well as by diminishing aggresssion-neutralizing influences. Economic hardships are frustrative conditions that activate anger and aggression. In a large sample study in three U.S. metropolitan areas, job loss was found to increase the risk of violent behavior by a sixfold ratio, controlling for age, alcohol disorder, other psychiatric disorder, gender, race, household SES, and previous violent behavior (Catalano, Dooley, Novaco, Wilson, & Hough, 1993). Violent behavior, during that investigation's 18-month study interval, increased the likelihood of job loss by a fifteenfold ratio, controlling for the above covariates. In a subsequent study involving a time-series analysis of regional economic data, communitywide job loss was found to affect the rate of psychiatric hospital civil commitments for behavior dangerous to others, controlling for autocorrelation and other types of commitments (Catalano, Novaco, & McConnell, 1997). This suggests a need for therapist sensitivity and advocacy regarding client employment issues.

Clearly, diminished economic resources aggravate anger difficulties, but in addition to the aversiveness associated with economic strain, anger dyscontrol negatively impacts important social relationships, especially marital and family support systems. Male Vietnam War veterans with PTSD, compared to theater veterans without PTSD, have been found to have severe family adjustment problems, including three times the rate of family violence, by both the veteran and the spouse/partner (Jordan et al., 1992). Those investigators also found that the spouses or partners of the veterans with PTSD reported high levels of nonspecific distress, and about half "felt on the verge of a nervous breakdown" (p. 923). Moreover, their children had a significantly greater likelihood of having clinical-range behavior problems. Trauma, anger, and violence not only diminish family support, but they also add additional stressful aggravations.

The economic and social resource deficits of aggression-prone populations are exacerbated by their lack of cognitive skills. The third reciprocity here is between intellectual functioning and aggressive behavior. Huesmann, Eron, and Yarmel (1988) have shown that aggressive behavior in childhood interferes with the development of intellectual functioning and is predictive of poorer intellectual achievement in adulthood, and that low intelligence makes the learning of aggressive response more likely. More generally, persons with clinical problems of anger and aggression often have cognitive skill deficits in areas fundamental to implementing cognitive-behavioral treatment. They frequently need help in elementary matters, such as identifying emotion, differentiating types and degrees of emotion, and recording self-observations, which are the foundation of self-regulatory procedures, such as self-monitoring. However, limitations in cognitive skills need not disqualify application of a cognitive-behavioral approach to anger disorder, as Benson, Rice, and Miranti (1986) and Black and Novaco (1993) have successfully applied cognitive-behavioral therapy anger treatment to mentally handicapped patients.

ASSESSMENT OF ANGER

Anger is a subjective emotion, and it is therefore very appropriate that anger has been assessed primarily by self-report procedures. However, the assessment of anger in a clinical context is highly reactive. Because of a variety of negative connotations and anticipated consequences of having a "high anger" designation, people will mask their anger reactions or their anger disposition on self-report assessments, whether it be on psychometric scales or on interviews. Clients with anger problems tend not to be at all forthcoming in self-disclosure, having a highly suspicious, distrusting mind-set. Thus, the interpretation of anger scores on self-report scales must be done cautiously. Establishing trust and confidentiality will maximize the validity of anger self-report measures. This issue is particularly salient with regard to forensic patients and others in secure settings, as well as when health, employment, or disability benefits might be affected by anger status.

Bearing in mind that anger can be assessed through a variety of clinical rating procedures, including interview and behavioral observation approaches, we here restrict our presentation to psychometric assessment of anger and to a few scales that we have found to be useful with regard to anger in the context of PTSD.

Two psychometric instruments for assessing anger by patient self-report are (1) the Spielberger State–Trait Anger Expression Inventory (STAXI; Spielberger, 1991) and (2) the Novaco Anger Scale (NAS; Novaco, 1994a). The STAXI includes measures of state anger, trait anger, and anger expression. The trait anger scale has subscales of angry temperament and angry reaction; the anger expression scale has subscales of anger-in, anger-out, and anger control. The STAXI has had extensive development and validation with adolescent and adult samples, from both normal populations and clinical populations with physical health problems (Spielberger, 1991). The STAXI combines the State–Trait Anger Inventory (STAI; Spielberger, 1988a) with the Anger Expression Scale (AX; Spielberger, 1988b).

The NAS has cognitive, arousal, and behavioral domain scales as components of its anger disposition measure. The NAS was developed and validated for use with mentally disordered and normal populations. In studies with psychiatric patients in California state hospitals (Novaco, 1994a), it was found to have an internal reliability of .95 and a test–retest reliability of .84, and to be significantly related to a number of anger and aggressive behavior criteria evaluated in concurrent, retrospective, and prospective analyses, which also included comparative anger measures. The concurrent correlation of the NAS scale with the Spielberger Trait Anger Scale was found to be .84; and its predictive correlation with Spielberger State Anger was found to be .36 at 2 weeks, .43 at 1 month, and .46 at 2 months with 151 patients in the analyses. As the NAS has theoretically specified subscales, it was designed to be an improvement over the Novaco Provocation Inventory (NPI; Novaco, 1975, 1988). The latter measure, previously 80 items in

length, has now been condensed to a 25-item scale, which measures anger reactions to hypothetical situations.

In PTSD research, the combined STAI and AX scales were used in the study of female crime victims by Riggs et al. (1992) and that of Vietnam War veterans by Lasko et al. (1994), both of which examined anger in PTSD and non-PTSD groups. The AX has been used in the laboratory study of anger, impulsivity, and PTSD by Chemtob et al. (1994), which also used the NPI. In the PTSD anger treatment study by Chemtob, Novaco, Hamada, and Gross (1997), the AX and the NAS were used. Pertinent to differentiating PTSD from non-PTSD groups, Riggs et al. found that State Anger and Anger-In were significant, Lasko et al. found all STAXI subscales to be significant, and Chemtob et al. (1994) found the AX and the NPI to be significant. The NAS has not yet been reported for a PTSD differentiating study, but we are presently engaged in that regard. A more extended discussion of anger measures pertinent to cognitive-behavioral treatment of anger can be found in Novaco (1994b).

Another potentially valuable index of anger are five items on the Mississippi Scale for Combat-Related Posttraumatic Stress Disorder (Keane, Caddell, & Taylor, 1988). These items, with their scale numbers in parentheses, are as follows: "If someone pushes me too far, I am likely to become violent" (3); "The people who know me best are afraid of me" (5); "I am frightened by my urges" (23); "I am an easygoing, even-tempered person" (27); and "I lose my cool and explode over minor everyday things" (31). We have found this set of anger/aggression items to have a high degree of internal consistency ($r = .87$) in a sample of 142 veterans referred for clinical services.[1] We have found this set of items, computed as an index, to be very significantly related to the AX and NAS measures.

ANGER TREATMENT AND PTSD

Given the far-ranging negative impact of anger problems, the dearth of research on anger treatment for PTSD patients is lamentable. Treatment intervention studies for PTSD have not been focused on, or even prioritized, anger as an outcome criterion. For example, Foa, Rothbaum, Riggs, and Murdock (1991) successfully treated female crime victims for PTSD with cognitive-behavioral interventions, but anger was not a specified treatment target in that study and was not part of the measurement protocol. Yet that patient population has been found to have significant anger in conjunction with PTSD (Riggs et al., 1992). Similarly,

[1]These five items are noted by Lasko et al. (1994) as being "aggression-related items" (p. 375), although they did not report separate analysis of them. We had already isolated this set of items as a subscale in our own research and computed the five-item total as an anger index with 77 patients in spring 1993. We are presently preparing an empirical report of the relationship of anger to PTSD based on our work with multiple samples of veterans.

in a PTSD treatment study with female sexual assault victims, Resick and Schnicke (1992) introduced their study making the point that PTSD is much more than a fear-based disorder and that intrusive memories and avoidance might be activated by strong affects other than fear. In that context, they assert that "crime victims often report experiencing anger" (p. 749). Curiously then, they did not describe any of their cognitive processing therapy as addressing anger, nor did they give any report of an anger measure, despite using the Symptom Checklist 90 (SCL-90) as a dependent variable and this scale having a hostility subscale. Research on male violence victims as well ignores anger in assessing PTSD symptoms, as did Burton, Foy, Bwanausi, Johnson, and Moore (1994) in their study of male juvenile offenders.

The scant attention given to anger treatment with PTSD populations may be a function of the strong priority given to fear and depression in conceptualizing PTSD symptomotology. However, the various obstacles to treating anger discussed earlier may also be significant in diverting focus. Given the range of impediments to doing therapy with seriously angry patients, our recent study with Vietnam War combat veterans having severe PTSD and severe anger offers an optimistic note for the efficacy of anger treatment. As the patients in our Vietnam War veteran study were very treatment-resistant, the results have implications for efficacy in treating the anger problems of other traumatized populations.

Despite the documented association between anger and combat-related PTSD, and despite the importance of treating the anger component of PTSD, there had been no empirically validated approach to anger treatment for this disorder. The project utilized a cognitive-behavioral intervention for anger that had been demonstrated to be effective with other clinical populations in research involving experimental, multiple baseline, and case study designs (Novaco, 1975, 1994b). In an important addition to the preexisting protocol (Novaco, 1983), we focused on the patients' cognitive schemas related to combat experience, threat, survival, and trauma as these structures affected their daily lives.

The treatment of anger remains a relatively neglected topic in clinical research, especially with seriously disturbed patients. Vietnam War combat veterans with PTSD most certainly fall into that latter category. The studies by Chemtob et al. (1994) and by Lasko et al. (1994) showed the heightened anger associated with Vietnam combat PTSD patients and called attention to the importance of anger control skills. These men are often remarkably treatment resistant and are additionally problematic because of their readiness to become angry during treatment and toward the therapist. Thus, our controlled study (Chemtob, Novaco, Hamada, & Gross, 1997) was an important step in advancing treatment implementation.

The cognitive-behavioral treatment of anger began with Novaco (1975) in an experimental study with an outpatient population. In that initial project, the anger treatment principally involved cognitive therapy and relaxation training applied in conjunction with graduated exposure to provocation. A treatment

components and control group design was used, and the combined anger treatment resulted in a significantly greater reduction in anger on multiple measures in multiple provocation modes, compared to a self-monitoring, attention control condition, and to the cognitive and relaxation component conditions.

The anger treatment was subsequently reconceptualized and developed further in terms of a stress inoculation framework (Meichenbaum, 1975), which emphasized cognitive-behavioral coping skills. The stress framework served to broaden the conceptualization of anger as a clinical problem and facilitated the presentation of the treatment as the learning of coping skills. Also, Meichenbaum's three-phase approach of cognitive preparation, skill acquisition, and application practice provided useful rubrics for the development of treatment components. The central components of the anger treatment were then constructed as cognitive restructuring, arousal reduction, and behavioral coping skills, and were successfully applied to a hospitalized patient with severe anger problems (Novaco, 1977a) and to police officers (Novaco, 1977b; Sarason, Johnson, Berberich, & Siegel, 1979). The training of therapists in the use of the treatment method was also experimentally validated in a controlled study with probation counselors (Novaco, 1980).

This stress inoculation approach to anger management has been evaluated with diverse clinical populations. Several experimental studies of this approach with institutionalized juvenile delinquents obtained significant treatment effects (Schlichter & Horan, 1981; Saylor, Benson, & Einhaus, 1985; Feindler, Marriott, & Iwata, 1984), and Feindler and Ecton (1986) developed some new treatment elements, including social skills training components. Other experimental studies of anger treatment based on the Novaco approach, using control and comparison groups, include Benson et al. (1986) with mentally retarded adults, Stermac (1983) with forensic patients, Moon and Eisler (1986) with college students, and Hazaleus and Deffenbacher (1986) with college students. Subsequent work by Deffenbacher gave explicit attention to arousal reduction in treating recruited college student clients (Deffenbacher, 1988; Deffenbacher, Story, Brandon, Hogg, & Hazaleus, 1988; Deffenbacher, Story, Stark, Hogg, & Brandon, 1987).

A number of case studies and multiple baseline design studies involving a variety of serious clinical disorders have provided further support for the efficacy of cognitive-behavioral interventions based on this anger treatment procedure. Successful treatment results have been reported by Nomellini and Katz (1983) with child-abusing parents, by Bistline and Frieden (1984) with a chronically aggressive man, by Spirito, Finch, Smith, and Cooley (1981) with an emotionally disturbed boy, by Lira, Carne, and Masri (1983) with a brain-damaged patient, by Black and Novaco (1993) with a mentally handicapped man, and by both Bornstein, Weisser, and Balleweg (1985) and Howells (1989) with institutionalized forensic patients. Recently, Renwick, Black, Ramm, and Novaco (1997) achieved significant treatment gains with very angry and assaultive psychiatric patients with serious mental disorder in a maximum security hospital.

This cognitive-behavioral approach to anger treatment involves the following key components: (1) client education about anger, stress, and aggression; (2) self-monitoring of anger frequency, intensity, and situational triggers; (3) construction of a personal anger provocation hierarchy, created from the self-monitoring data and used for the practice and testing of coping skills; (4) arousal reduction techniques of progressive muscle relaxation, breathing-focused relaxation, and guided imagery training; (5) cognitive restructuring by altering attentional focus, modifying appraisals, and using self-instruction; (6) training behavioral coping in communication and respectful assertiveness as modeled and rehearsed with the therapist; and (7) practicing the cognitive, arousal regulatory, and behavioral coping skills while visualizing and role-playing progressively more intense, anger-arousing scenes from the personal hierarchies.

Provocation is simulated in the therapeutic context by imagination and role play of anger incidents from the life of the client, as directed by the therapist. This is a graduated exposure based on a hierarchy of anger incidents produced by the collaborative work of client and therapist. This graduated, hierarchical exposure, done in conjunction with the teaching of coping skills, is the basis for the "inoculation" metaphor and is most central to the "stress inoculation" approach (cf. Meichenbaum, 1985).

This stress inoculation approach to anger control was the foundation of the specialized anger therapy implemented in our controlled treatment trial with Vietnam War veterans, which was conducted through the Hawaii VA, in collaboration with Roger Hamada, Doug Gross, and Gary Smith. Importantly, the anger treatment protocol was augmented by therapeutic derivations of the cognitive action model of PTSD and the concept of "survival mode" functioning discussed earlier. Centrally, in "survival mode," a person responds with context-inappropriate cognitive distortions that tend to confirm the presence of threat and lead to the defensive activation of anger and aggression. The shift into that mode of functioning is accompanied by a substantial loss of self-monitoring.

In augmenting the anger treatment protocol, therapists educated patients about the phase-shift into survival mode that occurs in PTSD and enabled them to recognize the phase shift dynamic and the automatic anger activation. Patients were helped to see that their anger was once functional as part of a survival response and was a legitimate attempt to adapt to that past survival context. With supportive guidance, they can then identify the present context inappropriate aspects of survival mode functioning and the dysfunctionality of the conjoined anger and aggression. The associated loss of self-monitoring can then be remedied.

Essential to reinstituting regulatory controls for anger and aggression is treating the central self-monitoring deficits. In that regard, the clinician helps the patient to (1) monitor the cognitions that he or she typically experiences when threatened and which induce anger episodes; (2) identify signs of arousal, including its intensity, duration, and lability in response to the perception of danger or

threat; (3) recognize the role that anger reactions play, both as responses to sensing danger and as behaviors that create danger for others, thus escalating the threat potential of a situation; and (4) distinguish impulsive actions from more controlled responses. The cognitive, arousal, and behavioral domains of anger are thus segmented for self-monitoring.

Anger has been commonly understood by patients and nonpatients, as being a "passion" by which one is "gripped," "seized," or "torn" (Averill, 1982), suggesting a loss of control. High intensity anger is patently distressing. Putting anger in a survival context and segmenting its domains of activation facilitate the development of regulatory skills, as its meaning is clarified and its intrinsic goals are validated. Instead of viewing anger as a mysterious force that takes charge of the personality, the client is given conceptual tools to make sense of his or her experience, to focus change efforts into partitioned subdomains of anger, and to gauge therapeutic progress more meaningly and realistically. The client is able to work on focused, goal-limited objectives, rather than on a global "anger problem" that can otherwise seem impenetrable and insolvable. This, then, provides a safeguard against the problem of frustration and demoralization discussed earlier as an obstacle to anger treatment. Correspondingly, the segmenting of anger domains enables the therapist to track therapeutic progress with greater sensitivity to change.

The cognitive distortions linked to threat perceptions and highly automatized anger have a powerful immediacy and validity. For war veterans with PTSD, the perceptual frame of reference is the combat environment, hence "what is" and "what was" are confused. Their day-to-day functioning occurs with a high degree of self-protectedness and reactivity. Life's hassles, insufficient resources, and postwar government administrative bureaucracy reinforce that reactivity. It is in this context that one encounters the continuance of threat perceptions and anger-engendering cognitions, such as "I'm expendable," "I'm a problem to them," "I can't let my guard down," "Anyone could be the enemy," and "If I screw-up, somebody dies." Being very much stuck in a self-protective mode, their cognitions are replete with dichotomous and polarized thinking (good–bad; friend–enemy). Even when the anger is disguised as flippancy or inappropriate laughter, it is not far from the surface and can be readily uncovered.

Anger is intrinsically infused with the theme of justification, and this is compounded for Vietnam veterans due to the well-known circumstances of the war. Indeed, anger became a core theme in group identity and in social cohesion connected to the traumatic experience, and it is fair to say that one risked group rejection by not sharing in justified beliefs of persecution and victimization. Anger expanded to intense moral indignation of group members against diffusely constituted "others"—saying, in effect, " 'They' are responsible for my actions," and, therefore, " 'They' can be discounted," and " 'They' betrayed me and abandoned me, and now I am entitled to exoneration and compensation." This strongly justified moral posture serves to entrench anger. Moreover, as "they" can be

discounted, one does not need to be concerned about "their" needs, desires, or worth. Hence, the fixation in the self-protective mode presents a formidable barrier to the role-taking perspective that is essential to anger regulation. Self-absorption, defensiveness, and preoccupation with threat run counter to empathic understanding when faced with an aversive experience. Rather than disappointment, one gets anger provocation.

Cognitive restructuring efforts must be grounded in the trust and safety of the therapeutic relationship. The "survival mode" concept provides a meaningful connection between the troublesome current anger eruptions and the originating traumatic exposure, which then facilitates consideration of alternative constructions of contemporary events, arousal modulation, and context-appropriate modes of responding. However, this is far from being a smooth road. For example, efforts to promote a shift to being empathic may import a confrontation with the trauma of have killed people, and resistance might then emerge (e.g., "If they don't care about me, why should I care about them?"). As a safeguard to ensure trust and promote therapeutic alliance, our recent controlled treatment trial used clinicians who were experienced therapists and also had extensive combat experience in Vietnam. Yet even this did not always help, as one veteran angrily denounced his therapist, who had been frequently in combat in Vietnam, saying, "Anyone knows that officers cannot be trusted." One can here see the refractoriness of anger-engendering schemas and the formidable obstacles to treatment engagement. Nevertheless, the project, which we next describe briefly, achieved significant treatment gains.

HAWAII VA TREATMENT STUDY

As the anger therapy is understood to be a treatment adjunct to routine psychological and medical care, the Chemtob, Novaco, Hamada, and Gross (1977) study randomly assigned patients to either (1) routine clinical care only, or (2) routine clinical care plus the specialized anger treatment, implemented over 12 sessions. The routine clinical care control condition consisted of an amalgam of treatments typically provided at veterans' centers and VA mental health clinics. These heterogeneous treatments consisted of psychiatric consultation, medication, and supportive psychological interventions, such as eclectic individual counseling, substance-abuse groups, and support/rap groups. Regarding the various psychological supportive interventions, each patient in the control group received at least two of these services, and several patients also received systematic desensitization addressing PTSD symptoms. Fifteen patients completed the assessment and treatment protocol, with 8 patients in the anger treatment condition and seven in the routine clinical care condition. In addition to pre- and posttreatment assessments, we conducted an 18-month follow-up for both treatment completers and dropouts.

The patients in the study had severe PTSD, reflected in their average Mississippi Scale score (M = 130.3; SD = 13.5), which exceeded by several standard deviations the cutoff score of 89.0 used in the National Vietnam Veteran Readjustment Study (Kulka et al., 1990) to discriminate combat-related PTSD. They were also significantly higher on this index than 170 consecutive admissions to a specialized Hawaii VA PTSD outpatient clinic (M = 118.6, SD = 23.7). The study participants were also an extremely angry group, as evidenced by their NAS scores (M = 112.7; SD = 15.8), which are substantially higher than California State Hospital civil commitment and forensic inpatients (M = 90.1; SD = 18.2) and also significantly higher than the PTSD clinic sample (M = 97.6; SD = 20.7).

Recognizing anger's multidimensionality, we measured (1) anger disposition, assessing trait-like aspects of anger responding, (2) anger reactions, pertaining to the impact of situational provocation, and (3) anger control, reflecting the capacity for anger regulation. Compared to the routine clinical care treatment condition and controlling for pretreatment scores, we obtained significant anger treatment group effects on multiple self-report measures of anger reactions and anger control at posttreatment, and the significant differences in anger control were maintained at 18-month follow-up. We also found in covariance analyses on posttreatment Clinician-Administered PTSD Scale (CAPS) indices that traumatic reexperiencing, in frequency and intensity, was significantly lower for patients in the anger treatment condition in contrast with the control group, and a similar effect was obtained for state anxiety. However, we did not find any significant anger treatment effects on physiological measures of anger reactions obtained in conjunction with imaginal provocations, nor did we find differential treatment group effects on anger disposition measures, although post hoc analyses did find a significant reduction from pretest to posttest in AX scores and NAS Cognitive scores for the anger treatment group. Overall, we found modest but noteworthy treatment gains for the anger treatment.

The size of our treatment groups is an obvious limitation of the study in evaluating its internal validity and generalizability. However, the routine clinical care condition represented a conservative control group, as it represented real treatment for PTSD, and we used conservative analyses and demonstrated strong effect sizes. An important cause for caution, though, is the high dropout rate that we experienced. Thirteen of the 28 patients who began treatment dropped out, representing an attrition rate of 46.4%. Reassuringly, treatment completers did not differ on age or education, nor on Mississippi or NAS scores, from treatment refusers or from treatment noncompleters, and we did not have differential dropout rates between treatment conditions.

In considering the attrition in our study, we compared the rate to that found in other studies. Riggs et al. (1992) studied anger in crime victims, taking measurements on two occasions 1 month apart. These investigators experienced a 26% dropout rate from one testing to the next in a sample scoring one standard deviation below ours in anger severity. Dropout rates in controlled treatment

studies (including inpatient samples, as well as psychopharmacological studies) of combat-related PTSD, as reviewed by Solomon, Gerrity, and Muff (1992), range from 25% with inpatients to 46% with outpatients. Our participants exhibited levels of PTSD symptomatology consistent with those of inpatients, and their retention was made more difficult by their outpatient status and by their extreme anger. Thus, our attrition rate was not unusual.

IMPLICATIONS AND FUTURE DIRECTIONS

Controlled studies of anger treatment have been done primarily with college students and with outpatients in college clinics. Our patients were severely angry, even when compared to incarcerated, violent forensic inpatients, This treatment population was extremely difficult to maintain and evaluate in a treatment outcome study context. Vietnam veterans with PTSD often experience substantial distrust of research, perceiving such efforts as exploitative, akin to their perception of exploitation during military service. The level of dropout that we experienced is likely to be characteristic of this severely disordered population.

We were well aware of the problem of treatment resistance regarding our patient population and constructed procedures intended to mitigate it. For example, we used experienced Vietnam veterans as therapists. We provided reminders of appointments, sought to establish therapeutic alliances with significant others, coordinated our treatment closely with other providers, and designed our treatment protocol to provide for primary care concurrent with our adjunctive treatment. These steps kept our dropout rates within the upper range experienced by other investigators with less severe PTSD samples, but it obviously did not resolve the problem of obtaining treatment engagement.

These issues also raise important questions with respect to the generalizability of our findings on anger treatment to similar patients outside the resources of a research project to other, less severely afflicted Vietnam veterans with PTSD, to persons with combat-related PTSD from other wars, or to other violence-related trauma. While questions about generalizability can only be resolved by further research, our clinical impression is that downward extensions of the protocol to other traumatized populations should prove fruitful.

Given that residual anger is commonly found among those who have experienced trauma and that this anger is associated with very problematic complications in personal adjustment, systematic investigation of anger assessment and anger treatment for PTSD ought to be undertaken. The nature of the relationship between PTSD and its symptom clusters to component dimensions of anger (e.g., cognitive, arousal, and behavioral) remains to be examined. However, by showing that patients with severe anger and severe PTSD can be helped to increase their anger regulatory abilities, our findings are clearly encouraging for continued work

with such populations and with other PTSD patients having great needs for clinical care. We believe that we have taken a worthwhile first step.

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